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Importance of maintained atrio-ventricular synchrony in patients with pacemakers

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KEY WORDS: Dual chamber pacemaker, single chamber pacemaker, AV delay, work capacity, morbidity, mortality, quality of life.

The effect of atrial contraction on cardiac function is reviewed in patients with dual chamber and rate-responsive ventricular pacemakers. The question posed was is there any haemodynamic, clinical or prognostic advantage of AV synchrony in dual chamber pacemakers in comparison to rate-responsive ventricular pacemakers? Optimal AV delay in dual chamber pacing favours cardiac performance at rest, while during exercise the increase in heart rate rather than AV synchrony influences cardiac performance and working capacity. However, there is little information on the benefit of maintained AV synchrony in patients' daily activities. Patients with pacemakers which maintain AV synchrony seem to have less morbidity and mortality than patients with ventricular stimulation alone, and there are comparable rates of complication in carriers of single and dual chamber pacemakers, the former showing problems with the pacemaker syndrome and the latter with atrial sensing and pacemaker-induced tachycardias. The disadvantage of dual chamber pacemakers are higher costs and time-consuming controls.

Introduction

In comparison with fixed rate ventricular pacing (VVI), rate-responsive pacemakers (VVIR) may increase cardiac output and exercise capacity by a rise in heart rate[1–5]. Dual chamber pacemakers (DDD) that sense the atrial wave, maintain the normal sequence of cardiac chamber activation (atrioventricular synchrony) and permit a chronotropic response to exercise in patients with normal sinoatrial function. During exercise, the additional atrial contribution to ventricular filling (atrial 'kick') in dual chamber pacing is supposed to improve haemodynamic function to a greater extent than in rate-responsive ventricular pacing alone. Any discussion about the importance of maintained atrioventricular (AV) synchrony has to answer the following: Is there any haemodynamic, clinical or prognostic advantage as regards AV synchrony in carriers of dual chamber pacemakers in comparison with rate-responsive ventricular pacemakers?

HAEMODYNAMIC ADVANTAGE OF MAINTAINED AV SYNCHRONY AT REST

Previous studies have shown that atrial contribution to ventricular filling in hearts with normal left ventricular function can cause a more than 20% increase in cardiac output[6–13]. Particularly susceptible to this improvement are those patients with left ventricular hypertrophy[14,15], those of advanced age[16] or with left ventricular diastolic dysfunction[17,18]. However, controversy exists about patients with impaired left ventricular systolic function. Several studies proved the value of AV synchrony in these patients[18–20] but they did not evaluate the influence of left ventricular filling pressure on atrial contribution to cardiac output. Greenberg *et al.*[21] and Myreng *et al.*[22] found an inverse relationship between ventricular filling pressure and atrial contribution to cardiac output. Faerestrand and Ohm[6], French *et al.*[23] and Dritsas *et al.*[24] found no benefit in relation to atrial systole in patients with left ventricular failure. Consequently, most evidence is demonstrated in hearts with normal ventricular filling pressures. Thus, at high passive filling pressures the ventricle is maximally dilated and operates at the extreme of the flattened Frank–Starling curve. Therefore, a further increase in preload by the atrial 'kick' does not augment stroke volume.

Irrespective of the underlying cardiac disease, in order to maximize cardiac output in patients with dual chamber pacemakers it is essential to have an appropriately timed atrial contraction prior to the onset of ventricular systole. The timing of atrial contraction is important in coordinating AV valve closure, since the major mechanism of such a closure is the increase in ventricular end-diastolic pressure[18]. An inappropriately timed AV delay may cause mitral regurgitation and impaired diastolic filling[25]. To avoid unnecessary ventricular pacing and early battery depletion, the AV delay is often lengthened to achieve an intrinsic ventricular contraction, but an impaired haemodynamic function has also been demonstrated in unpaced patients whose PR intervals are too long[26,27].

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Table 1 Optimal AV delay in patients at rest

Reference, year	Parameters measured	Optimal AV delay (ms)	Remarks
Nitsch[9], 1984	SV, CO	100–150	—
Mehta[28], 1989	CO	150	—
Iwase[29], 1986	LVIV	150	—
Wish[31], 1987	SV	144	200 ms for long interatrial delays
Janosik[32], 1989	CO	144 or 176	144 ms for VDD, 176 ms for DVI pacing
Frielingsdorf[33], 1994	EF	150, 200	—
Sulke[34], 1992	Quality of life	125, 175 RR	—
Haskell[35], 1986	CO	150, 200	150 ms for fast heart rates
Videen[36], 1986	EF, CI	140–150	190–200 ms for patients with reduced EF
Dritsas[24], 1993	SV	172, 142, 97	172 ms for diastolic dysfunction, 97 ms for systolic dysfunction, 142 ms for normal LV function

AV=atrioventricular, CO=cardiac output, CI=cardiac index, DVI=AV sequential pacing, EF=ejection fraction, LV=left ventricular, LVIV=left ventricular inflow volume, RR=rate responsive AV delay, SV=stroke volume, VDD=atrial triggered ventricular pacing.

Even deviations in AV delay, by as little as 25 ms from an individual's optimal AV delay, resulted in a significant decrease in cardiac output[26,28–32]. In a study by our group[33] some patients showed a higher ejection fraction with single chamber ventricular pacing than with dual chamber pacing with an inappropriately timed AV delay (Table 1). Therefore, an inappropriately timed atrial systole as a result of a dual chamber pacemaker may be of little or no benefit as regards the haemodynamic effect than a ventricular-inhibited single chamber pacemaker[9,13]. Little has been reported on the clinical relevance of optimizing the AV delay. Sulke *et al.*[34] found that short AV delay settings (125 ms, 175 ms) were better tolerated than longer settings (250 ms) during out-of-hospital activity.

AV delays between 150 to 200 ms produced maximal left ventricular ejection fraction (Table 1). The optimal AV delay which makes atrial contraction coincide with late diastole and thus contribute most to left ventricular filling[29] is determined by factors such as heart rate[28,35], left ventricular function[24,36–38], atrial size[39] and the inter-individual variability of the intra-atrial conduction time[31,32,40].

A shorter or longer intra-atrial conduction time corresponds to a shorter or longer AV delay[31]. This is of special clinical relevance when pacing mode changes from dual chamber sequential (DVI) to atrial triggered ventricular pacing (VDD) because the left atrial to left ventricular sequence increases. To avoid a change in timing of left atrial contraction, with resulting decrease in stroke volume, the programmed AV delay must be shorter during atrial-triggered than during dual chamber sequential stimulation[31,32,36]. A difference of 30–50 ms may be necessary to optimize cardiac output[31,32,41] which emphasizes the importance of the AV delay hysteresis with altering pacing modes. This plays a particular role in the sick sinus syndrome which requires frequent atrial pacing.

Patients with hypertrophic cardiomyopathy or long-standing hypertension have abnormal ventricular diastolic function. The apparent increase in muscle stiffness and associated resistance to passive diastolic filling of the left ventricle underline the importance of an appropriately timed atrial systole in the preservation of ventricular filling in late diastole[37,38]. Shefer *et al.*[17] demonstrated striking haemodynamic differences between ventricular and AV sequential pacing. Loss of atrial synchrony was associated with a symptomatic fall in stroke volume. Dritsas *et al.*[24] observed that optimizing the AV delay is more important in patients with impaired left ventricular diastolic function than in patients with severely impaired systolic function. Therefore, efforts should be made to maintain AV synchrony in patients with abnormal diastolic function, e.g. hypertrophic cardiomyopathy, and, thus, an AV sequential pacemaker should be used.

HAEMODYNAMIC ADVANTAGE OF MAINTAINED AV SYNCHRONY DURING EXERCISE

Rate-adapted AV delay pacing

Whether a rate-adapted or a fixed short AV delay improves haemodynamic performance during exercise remains controversial (Table 2). A significant effect[28,42,43] or no effect at all[44–46] on haemodynamics or clinical parameters has been reported in patients with a shorter AV delay during exercise than at rest. The differing results might be explained by different study designs, especially when exercise capacity was only submaximal or maximal. Sheppard *et al.*[47] investigated patients with complete AV block and found that at lower exercise levels cardiac haemodynamics did not differ when pacemakers were programmed to rate-adapted or fixed AV delays. At peak exercise, however, patients randomized to a rate-adapted AV delay showed better cardiac haemodynamics than patients with a fixed

Table 2 Benefit of an optimized AV delay during exercise

Reference, year	Parameters measured	Method used	Remarks
With benefit			
Mehta[28], 1989	CO	Doppler	AV delay of 75–100 ms preferred to AV delay of 150–200 ms
Leman[42], 1985	EF, SV	RNV	AV delay of 100 ms preferred to 150 ms
Ritter[43], 1989	CI, SVI	Thermodilution	Rate- adapted AV delay preferred to a fixed AV delay (150 ms, 200 ms)
Without benefit			
Lau[44], 1990	CO	Doppler	Same CO for short (75 ms) and long 150 ms) AV delays
Haskell[45], 1989	Exercise capacity	0_2 uptake	Same exercise capacity for short (66 ms) and long (168 ms) AV delays
Ryden[46], 1988	Exercise capacity	O_2 uptake	Same exercise capacity for various AV delays (50, 100, 200, 250 ms)

AV=atrioventricular, CI=cardiac index, CO=cardiac output, EF=ejection fraction, RNV=radionuclide ventriculography, SV=stroke volume, SVI=stroke volume index.

AV delay, although the duration of exercise was the same. The atrial contribution to ventricular filling is minimized or absent depending on the sensing of the P-wave, i.e. at higher heart rates induced by heavier exercise the P-wave coincides with the previous T-wave and, thus, eliminates the atrial 'kick'. Irrespective of the haemodynamic benefit, a rate-adapted AV delay seems to be preferable to a fixed AV delay which allows the upper tracking rate to increase with less limiting upper rate behaviour.

Dual chamber versus rate-adapted ventricular pacing

The same controversy exists whether dual chamber pacing is better than rate-responsive ventricular pacing. While some investigators[8,44,48–50] demonstrated a slight but significant improvement in cardiac output during exercise due to AV synchrony, others[23,46,51–54] entirely attributed this improvement to the heart rate increase and not to AV synchrony (Table 3). This discrepancy is probably a matter of different pacemaker settings, posture, work loads, study populations and underlying heart disease. Lemke *et al.*[55] found in patients with isolated sick sinus node disease a more pronounced increase in oxygen consumption and work capacity during rate-responsive dual chamber pacing than during rate-responsive ventricular pacing. In patients with a high degree of AV block the differences between the pacing modes were not significant. Furthermore, the distinction was rarely made between patients with normal and impaired left ventricular function. French *et al.* demonstrated[23] in patients with reduced left ventricular function, that during exercise physiological AV synchrony is less important that rate-responsiveness. During exercise the shortening of diastole unfavourably adds to high left ventricular filling pressures in the diseased heart; thus, the beneficial effects of AV synchrony may be even less. In a population with normal left ventricular function Ausubel *et al.*[56] showed that atrial synchronous pacing results in enhanced ventricular filling and spared contractile reserves when compared with rate-responsive ventricular pacing, but heart rate, blood pressure and work load remained the same during exercise in the two pacing modes. In our own study[50] none of the patients with normal or with impaired left ventricular function benefited clinically from a preserved AV synchrony; however, patients with normal left ventricular function and dual chamber pacing showed a higher oxygen consumption. Some of the discrepancies between the quoted studies may be accounted for by differences in exercise, which was either moderate or symptom-limited. Linde-Edelstam *et al.*[57] found that the relative contribution of atrial systole to ventricular filling is most important at rest, and that during exercise the relative contribution of atrial systole diminished concomitantly with a rise in mitral blood flow velocity. Thus, increasing kinetic energy of blood flow reduces atrial contribution to left ventricular filling.

QUALITY OF LIFE AND MAINTAINED AV SYNCHRONY

The effects of pacing mode on quality of life has been less studied. While Oldroyd *et al.*[58] and Bubien and Kay[59] failed to find any difference in symptoms between dual chamber and rate-responsive ventricular pacing, others[60–64] demonstrated a better quality of life with dual chamber pacing. Linde-Edelstam *et al.*[61] explained the patients' preference for dual chamber pacing in comparison with rate-responsive ventricular pacing by a significant reduction in cardiovascular symptoms, improved self-perceived health, enhanced physical ability and psychological well-being. However, mean heart rate during the active hours of the day was higher with dual chamber pacing than with rate-responsive ventricular pacing. Menozzi *et al.*[62] investigated 14 patients with high degree AV block and found that the improvement in quality of life was reflected by the haemodynamic advantages of atrial synchronization. Five of these 14 patients did not tolerate rate-responsive ventricular pacing and were switched to dual chamber pacing because of intolerable symptoms, such as signs of

Table 3 Importance of pacing mode during exercise: VVIR versus AV synchronous (DDD, VDD) pacing

Reference, year	Parameters measured	Method used	Remarks
AV with benefit			
Karlöf[8], 1975	CO	CVC, Fick	—
Lau[44], 1990	CO	Doppler	-
Lemke[48], 1990	SV, ANP	CVC, Fick	ANP levels were equal
Landzberg[49], 1990	Exercise capacity	Treadmill (time)	-
Frielingsdorf[50], 1993	Exercise capacity	O_2 uptake	patients with normal EF
AV without benefit			
French[23], 1988	Exercise capacity	0_2 uptake	Patients with reduced EF
Ryden[46], 1988	Exercise capacity	O_2 uptake	—
Frielingsdorf[50], 1993	Exercise capacity	O_2 uptake	Patients with reduced EF
Kristensson[51], 1985	SV, CO	Dye dilution	—
Pehrson[52], 1983	Exercise capacity	Bicycle (Watt)	—
Fananapazir[53], 1983	Exercise capacity	Treadmill (time)	—
Linde-Edelstam[54], 1992	Exercise capacity SV, CO	O_2 uptake CVC, Fick	Epinephrine and norepinephrine levels were equal

ANP=atrial natriuretic peptide, AV=atrioventricular, CO=cardiac output, CVC=central venous catheter, DDD=dual chamber pacing, SV=stroke volume, VDD=atrial triggered ventricular pacing, VVIR=rate-adapated ventricular pacing.

congestive heart failure and palpitations. These five patients were compared to nine patients who did not require the change to dual chamber pacing. The five patients were characterized by a lower incidence of organic heart disease and smaller echocardiographic chamber diameters. Sulke *et al.*[63] demonstrated that patients who preferred rate-responsive dual chamber pacing but felt that rate-responsive ventricular pacing was least acceptable had greater increases in stroke volume when paced in dual chamber mode than in the ventricular inhibited mode. Five out of 22 patients demanded early crossover because of intolerable symptoms, such as dyspnoea, dizziness and palpitations. Mitsuoka *et al.*[64] who investigated both patients with the sick sinus syndrome and retrograde atrioventricular conduction and those with high degree AV block, demonstrated beneficial effects on symptoms of dual chamber pacing compared with ventricular pacing.

The weakness of most studies which deal with quality of life is that AV delay is not appropriately timed and the programming of rate-responsive parameters is not optimized. A programmed activity sensor that is too sensitive with inappropriately high heart rates has been demonstrated to be poorly tolerated by patients[65].

Sulke *et al.*[66] reported that most patients who were satisfied with long-term VVI pacing benefited from the upgrading of ventricular to dual chamber pacing; this suggests there is a 'subclinical' pacemaker syndrome. The disadvantage of ventricular pacing is related to the absence of — or random occurrence of — atrial contribution to ventricular filling; thus, stroke volume is especially decreased at rest. A further unfavourable factor is the presence of retrograde atrial contraction (VA conduction) which occurs simultaneously or just after the paced ventricular contraction. The lack of coordination between atrial and ventricular contraction may provoke palpitations and hypotension with symptoms of dizziness which have been described as the 'pacemaker syndrome'[67]. The atrial contractions against closed AV valves, which induce retrograde atrial emptying and atrial distension, may initiate unfavourable peripheral autonomic reflexes[68 70] or blunt the baroreceptor reflex; cardiac output sometimes decreases to levels lower than during unpaced sinus bradycardia. Patients with the sick sinus syndrome are particularly prone to develop the pacemaker syndrome because they often have preserved AV conduction. Up to 90% of such patients may have a VA conduction[71], whereas the cumulative incidence of the pacemaker syndrome has been reported as high as 7% and as low as 0·5%[72,73]. The haemodynamic compromise observed with VA conduction appears disproportionately greater than would be expected if it were the mere consequence of the loss of atrial transport. VA conduction may cause atrial contraction to coincide with a closed AV valve and produce pronounced atrial distention. Nishimura *et al.*[74] demonstrated that nearly all patients with symptomatic ventricular-inhibited pacing-induced hypotension had intact VA conduction. Another entity is the 'AAIR pacemaker syndrome'[75,76]. Patients chronically paced

Table 4 Atrial fibrillation, chronic heart failure and mortality in ventricular (VVI) versus atrial based pacing (AAI, AV synchronous) in patients with the sick sinus node syndrome or AV block

Reference, year	Follow-up (months)	Patients (n)		AF (%)		CHF (%)		Mortality (%)	
		VVI	AAI/AV	VVI	AAI/AV	VVI	AAI/AV	VVI	AAI/AV
Sick sinus node									
Stangl[77], 1990	53	112	110	19	6	—	—	31	19
Feuer[78], 1989	44	70	61	25	11	—	—	—	—
Rosenqvist[80], 1986	24	79	89	30	4	23	7	10	6
Markewitz[81], 1986	32	87	136	30	7	—	—	—	—
Santini[83], 1990	54	125	214	47	7	—	—	30	14
Rosenqvist[84], 1988	48	79	89	47	7	37	15	23	8
Sasaki[85], 1991	62	34	41	44	17	21	2	24	0
Zanini[84], 1989	40	57	53	18	4	5	2	18	9
Sasaki[87], 1988	35	25	24	36	0	28	4	24	13
Andersen[88], 1993	*	115	110	19	10			13	11
Albert[93], 1987	60	79	49	—	—	—	—	26	16
Mean**				28	7	24	6	24	12
AV block									
Alpert[94], 1986	60	132	48	—	—	—	—	34	15
Linde[95], 1992	65	74	74	—	—	—	—	36	32
Mean								35	25

AAI=atrial pacing, AV=atrioventricular synchronous pacing, AF=atrial fibrillation, atrial flutter, CHF=congestive heart failure, *no data; **study no. 80 was excluded because it was amalgamated with no. 84

in the rate-responsive atrial mode may have a paradoxically increased stimulus-R interval, producing P waves occurring immediately after, or even within, the R wave of the preceding cycle which nullifies the haemodynamic contribution of the atrial systole. This may result in a limitation of the exercise capacity.

MORBIDITY AND MORTALITY WITH AND WITHOUT MAINTAINED AV SYNCHRONY

Morbidity: Atrial fibrillation, thromboembolic complications and chronic heart failure (Table 4, Fig. 1)

In patients with a sick sinus node disease the incidence of a new onset of atrial fibrillation, with its associated thromboembolic complications, is higher with ventricular stimulation (VVI) than with a stimulation in which AV synchrony is preserved (atrial based pacing: AAI, VDD, DDD). The review of several studies[77–88] reveals an incidence of atrial fibrillation of 18–47% among patients with ventricular stimulation and 0–17% of all patients with AV synchronous stimulation. A literature review by Sutton and Kenny[89] demonstrated over a period of 31 months a 12·3% incidence of thromboembolic complications in patients with ventricular stimulation (n=347) in comparison with 1·6% ($P<0·001$) with atrial stimulation (n=321). The incidence of chronic heart failure for both ventricular and AV synchronous pacing resembles the corresponding incidence of atrial fibrillation[84–87] for these pacing modes. There are no data available on patients with high degree AV block and pacing modality as regards atrial fibrillation or chronic heart failure.

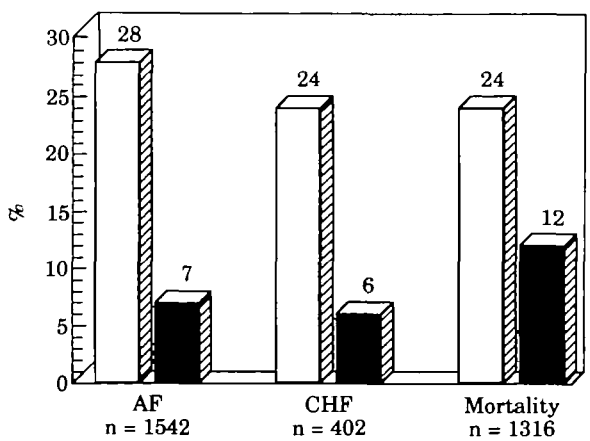

Figure 1 Percentage of chronic atrial fibrillation/flutter (AF), chronic heart failure (CHF) and mortality with ventricular (VVI, □) and atrial based (AAI/AV, ■) pacing, reported in patients with the sick sinus node disease (average of Table 4). The percentage of patients is given on top of each bar, n=total number of patients.

Mortality (Table 4, Fig. 1)

The 5-year survival rate of patients with a sick sinus node disease is, on the one hand, comparable with the mortality of patients with high degree AV block, and on the other with mortality of the non-paced population[90–92]. The prognosis for survival is related to the underlying heart disease and left ventricular function. The influence of pacing modality on survival has been addressed, in particular, in patients with sinus node disease and it is the object of only a few studies[77,83 88,93]. Rosenqvist *et al.*[84] reported a mortality of 23% in the population with ventricular stimulation in comparison with the significantly lower mortality of 8% in the atrial paced population. Alpert *et al.*[93] demonstrated a lower survival rate in patients with chronic heart failure — but not in patients without heart

failure — and ventricular stimulation than in patients with dual chamber stimulation. In patients with high degree AV block, AV synchronous pacing was preferable to ventricular pacing only when congestive heart failure was present[94,95].

The quoted studies demonstrate the superiority of AV synchronous pacing to ventricular pacing. However, some critical remarks are necessary: the conclusions of these studies only apply to paced patients with a sick sinus node disease. Patients with high degree AV block were not sufficiently investigated. Apart from two exceptions[85,88], all data originate from retrospective rather than randomized prospective trials. In some studies the study population was small and patients with fixed-rate ventricular stimulation served as controls. There are only limited data available which compare rate-responsive ventricular with AV synchronous stimulation. Van Erckelens *et al.*[82] showed a similar incidence of atrial fibrillation with rate-responsive ventricular and dual chamber pacing.

In patients with the sick sinus node disease and without AV conduction abnormalities, atrial inhibited pacing may be an alternative to dual chamber pacing. A study by Elshot *et al.*[96] with a follow-up of 12 years revealed an annual incidence of permanent atrial fibrillation of 1% which is comparable to the results of other recent investigations[97,98]. In the same study[96] the annual incidence of chronic atrial fibrillation in atrial pacing was reported from several other studies to range between 0·4 and 3·3%, compared to 4·3 and 11·7% in ventricular pacing; the reported annual mortality rate was between 0·5 and 4·8% in atrial pacing compared to 3·5 and 9·3% in ventricular pacing. Chronic atrial fibrillation mainly develops in patients with the brady-tachy syndrome[96] or in patients with a long history of episodes of pre-implant paroxysmal atrial fibrillation lasting more than 1 h[99]. Atrial inhibited pacing in the sick sinus node disease may be limited by progression of high degree AV block, but two reviews of 28[89] and 27 studies[100], respectively, showed a low annual incidence of AV conduction disturbances ranging between 0·7 and 3%.

Atrial inhibited pacing might even be superior to dual chamber pacing, partly due to the prevention of bradycardia-induced inhomogeneity of atrial refractoriness and partly to improved haemodynamics. There is evidence that atrial pacing may prevent paroxysmal atrial fibrillation[101,102]. Haemodynamic characteristics are better with atrial-inhibited pacing compared to dual chamber pacing[103–105] which is partly explained by intrinsic AV conduction[106] and synchronous ventricular contraction[107,108]. To our knowledge, there are no prospective morbidity and mortality studies comparing atrial inhibited pacing to dual chamber pacing.

COMPLICATIONS AND COSTS

Complications

The implantation of a dual chamber pacemaker is not associated with a higher rate of complications than that of a single chamber pacemaker. Whereas the most frequent complication of ventricular stimulation is the pacemaker syndrome, the dual chamber pacemaker is prone to malfunction of atrial sensing and pacemaker-induced tachycardia. Mueller *et al.*[109] prospectively analysed 258 patients with single chamber (VVI) and 75 patients with dual chamber pacemakers over a period of 3 years. There was no significant difference in relation to reintervention (5% of single chamber and 5·3% of dual chamber pacemakers), lead displacement with reoperation (1% vs 1·3%), infection (0·77% vs 1·33%), muscular stimulation (3·5% vs 4%) and urgent reprogramming (9% vs 8%). Goldman *et al.*[110] showed a higher perioperative complication rate in patients with dual chamber pacemakers (6·8%) than in patients with single chamber pacemakers (2·6%). In the study of Parsonnet *et al.*[111] there was a substantially larger incidence of complications when the implanters performed less than 12 implantations per year, this was particularly true for the incidence of lead-related complications.

Costs

Over a 12-year period Eagle *et al.*[112] compared the incremental cost of single and dual chamber pacemakers. At implantation, the single chamber device costed US $2503 less than the dual chamber device ($6924 compared with $9427) and after 12 years the cost difference was $5167 ($11 339 compared with $16 506). Thus, the difference in cost between single and dual chamber pacemakers is not simply a reflection of the initial implantation costs. The cumulative difference in costs continues to increase with time, and the major contribution was the more frequent failure rate of dual chamber generators. In this 12-year frame, a recipient of a dual chamber pacemaker would anticipate 0·74 generator replacements compared to 0·46 for single chamber pacemaker recipients. The differences would have been even greater if patients had survived longer. The quoted study was published in 1986; thus, as a result of improved survival in dual chamber generator batteries a third of the eventual cost difference could be eliminated. However, one of the major contributors to post-implantation costs, which remained unchanged over time, was the more frequent follow-up schedule for patients with dual chamber devices[112]. In the case of single chamber ventricular devices, the pacemaker syndrome and its resulting need for pacemaker revision to a dual chamber system accounted for just a small fraction of total cost[112]. A recent British study[113], which covered Northern England, with a population of three million inhabitants, showed considerable changes in regional pacing practice. The adoption of the optimal pacing practice recommended by the British Pacing and Electrophysiology Groups, e.g. dual chamber instead of single chamber pacing, would result in a substantial increase in expenditure. According to these recommendations the annual pacing budget for pacing hardware would have increased from £333 535 to £647 163 (increase of 94%) for the use of the optimal pacing mode. But these studies have not taken into account lower

morbidity and mortality associated with dual chamber pacing, resulting in a considerable health benefit, thus, reducing rehospitalizations and costs. There might be a substantial reduction in costs if atrial pacemakers were more widely used than dual chamber systems in patients with a sick sinus node disease. Costs could also be saved by changing a single chamber atrial pacemaker to either a ventricular pacemaker (because of chronic atrial fibrillation) or to a dual chamber pacemaker (because of progression of AV conductance disturbances).

Conclusions

The effect of atrial contraction on cardiac function is complex and depends on different interacting factors, such as left ventricular systolic and diastolic function, intra-atrial conduction time as well as atrial size and contraction. It seems reasonable to conclude that dual chamber pacing is superior to ventricular pacing, as regards haemodynamics at rest and quality of life. The data on optimal pacing during exercise conditions would require more homogeneous study designs for the determination of the importance of maintained AV synchrony, such as work loads, optimized AV delays and study populations. Furthermore, it remains to be determined whether other groups such as older patients, less active patients or those with diastolic dysfunction may especially benefit from AV synchrony. Patients with pacemakers which maintain AV synchrony seem to have a lower morbidity and mortality than patients with single chamber ventricular stimulation, but larger prospective randomized trials are needed to prove or disprove this hypothesis.

Atrial inhibited pacing is an under-used mode of cardiac stimulation but still remains the best pacing mode in many patients with the sick sinus node disease, whereas dual chamber pacing is the preferred mode in patients with high degree AV block. Today, single chamber ventricular pacing is restricted to patients with chronic atrial fibrillation and brady-arrhythmias.

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